The Neurologist: Clinical & Therapeutics Journal

Albert, Neurol Clin Therapeut J 2023, 7:4

Mini Review Open Access

Understanding Alzheimer's Disease: Unraveling the Mysteries of Memory Loss

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Abstract

Dementia, which is characterized by a decline in thinking and independence in daily tasks, is mostly brought on by Alzheimer's disease (AD), a sickness that results in the degradation of brain cells. The cholinergic and amyloid hypotheses were put up as two key causes of AD, and AD is thought to be a complex illness. The condition is also influenced by a number of risk factors, including as advancing age, hereditary factors, head injuries, vascular diseases, infections, and environmental variables. Only two kinds of medications are now approved to treat AD, cholinesterase enzyme inhibitors and N-methyl d-aspartate antagonists. These medications only work to treat the symptoms of AD; they do not treat the underlying cause of the disease.

Keywords: Memory loss; Cognitive decline; Neurodegenerative disorder; Amyloid plaques; Tau tangles; Brain atrophy

Introduction

The maximum regular shape of dementia, Alzheimer's ailment, is known as after the German psychiatrist Alois Alzheimer and is characterised via way of means of neuritic plaques and neurofibrillary tangles because of amyloid-beta peptide buildup within side the mind's maximum affected region, the medial temporal lobe and neocortical structures. When Alois Alzheimer tested the mind of his first patient, who skilled reminiscence loss and an extrade in persona earlier than passing away, he determined the presence of amyloid plaques and a widespread lack of neurons. He described the infection as a horrible ailment of the cerebral cortex. In his psychiatric handbook's 8th edition, Emil Kraepelin for the primary time cited this infection as Alzheimer's ailment [1].

The maximum regular shape of dementia is Alzheimer's ailment, which impacts as a minimum 27 million people international and bills for 60 to 70% of dementia cases. In addition to having a widespread monetary burden on society, the presence of this circumstance has a widespread have an effect on at the patient's families high-satisfactory of life [2]. From an anatomopathological perspective, AD is outstanding via way of means of prototypical lesions:

- Senile plaques, which might be extra-mobile lesions made from a nucleus of -amyloid protein accumulation and
- Neurofibrillary tangles, which might be intraneuronal findings made from phosphorylated tau protein. Amyloid cerebral angiopathy, which ends within side the deterioration of vascular wall additives and impairment of blood flow, also can be added on via way of means of the deposition of -amyloid protein in capillary walls, arteries, and arterioles.

The genetic aspect of Alzheimer's ailment seems to be due to an autosomal dominant mutation in one of the presenilin genes on chromosomes 1 and 14 or the amyloid precursor protein gene on chromosome 21. In addition, human beings with Down syndrome are at an extended hazard for early onset AD [3]. Although the genetics of AD are extra complicated and much less understood. It is thought that the epsilon 4 allele of the apolipoprotein E gene positioned on chromosome 19 is a hazard aspect for the improvement of sporadic AD [4].

Studies have proven that decrease stages of nutrition D are related to all varieties of dementia and Alzheimer's ailment [5]. The energetic shape of nutrition D, 1, 25-dihydroxyvitamin D3, regulates the expression of neurotrophins, along with nerve boom aspect, neurotrophin 3, and glial-derived neurotrophic aspect, in addition to neuronal survival, improvement, and function. In vitro, nutrition D will increase the phagocytic elimination of amyloid plaques via way of means of stimulating macrophages [6].

Various research has proven that nutrition D may be neuroprotective and about 50 nmol/L is enough for dementia hazard. These statistics are beneficial for designing cost-powerful randomized managed trials to research whether or not nutrition D supplementation can be useful in delaying or stopping the onset of dementia and AD in older adults [7].

Physiology of Alzheimer's disease

The telltale symptoms and symptoms of AD are extracellular plaques of the insoluble peptide -amyloid and neurofibrillary tangles of P-tau within side the cytoplasm of neurons. The deposits are concept to reason atrophy and demise of neurons because of excitotoxicity processes, fall apart in calcium homeostasis, inflammation, and depletion of electricity and neuronal factors. Although the mechanisms through which those modifications result in cognitive decline are nevertheless below debate [8]. The aforementioned cognitive loss is because of this process, which damages neurons and synapses concerned in reminiscence processes, learning, and different cognitive activities. One of the maximum broadly identified thoughts approximately the pathophysiology of AD, the amyloid cascade theory, despite the fact that nevertheless debatable, states that the cerebral accumulation of A-peptide.

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Received: 05-Aug-2023, Manuscript No: nctj-23-109323, **Editor assigned:** 07-Aug-2023, PreQC No: nctj-23-109323 (PQ), **Reviewed:** 21-Aug-2023, QC No: nctj-23-109323, **Revised:** 24-Aug-2023, Manuscript No: nctj-23-109323 (R), **Published:** 31-Aug-2023, DOI: 10.4172/nctj.1000150

Citation: Albert S (2023) Understanding Alzheimer's Disease: Unraveling the Mysteries of Memory Loss. Neurol Clin Therapeut J 7: 150.

Risk factors

AD may be labeled primarily based totally on whilst the primary signs and symptoms appeared. About 4-6% of instances of AD are early-onset instances, which have an effect on the ones below sixty five. Late-onset AD, on the opposite hand, impacts human beings sixty five and older. In addition to the age at which signs and symptoms first appear, there also are clinical, cognitive, neuropathological, and neuroimaging variations among the early and past due varieties of AD [9]. About 70% of the hazard of obtaining AD may be ascribed to genetics. Early Alzheimer's disorder is commonly as a result of mutations within side the APP, PSEN1 and PSEN2 gene, however past due-shape AD is frequently connected to an APOE gene polymorphism [10].

Causes

One theory is that plaques prevent nerve cells in the brain from communicating properly. Confusion can make it difficult for cells to get the nutrients they need. It is understood that as Alzheimer's develops, certain nerve cells die, and as the disease progresses, more and more nerve cells, also called neurons, are lost [11].

- Age: Age is the most important factor in the development of Alzheimer's disease. The chance of developing the disease doubles every five years after age 65.

- Down syndrome: This is because the genetic defect that causes Down syndrome can cause amyloid plaques to build up in the brain over time, which can lead to Alzheimer's disease in some people.

- Genetics: Based on twin and family studies, the genetic heritability of Alzheimer's disease varies between 49 and 79 percent. About 0.1% of cases are autosomal (non-sex related) dominant inherited forms that occur before the age of 65. This form of the disease is called early onset familial Alzheimer's disease. Although rare, a small percentage of people develop AD before age 65. Three genes involved in causing AD due to these mutations are amyloid precursor protein, presenilin 1 and presenilin 2 [12].

Future aspects

Diseases such as Alzheimer's require early diagnosis for effective treatment. As the number of Alzheimer's cases is increasing at an alarming rate, it is imperative to use advanced technology to fight this disease. In recent years, many researchers have done and continue to study biomarkers, proteomics and genomics. Despite these studies, there are still several challenges to overcome. The availability of technology alone will not help control the disease, standardization of methods and techniques is paramount to maintain consistency and achieve essential reliability.

Discussion

Alzheimer's disease is a neurodegenerative disorder characterized by the accumulation of abnormal proteins, beta-amyloid plaques, and tau tangles in the brain. These aggregates disrupt neuronal communication and lead to cognitive decline, particularly affecting memory and executive functions. Research has identified several risk factors for Alzheimer's, with age being the most significant one. Genetic factors and family history also play a role, along with lifestyle choices and cardiovascular health. While the disease cannot be entirely prevented, adopting a healthy lifestyle, including regular physical activity, a balanced diet, mental stimulation, and social engagement, may help reduce the risk or delay its onset. Diagnosing Alzheimer's can be challenging, especially in the early stages when symptoms may be subtle. Currently, a definitive diagnosis can only be made postmortem through brain examination. However, advances in neuroimaging and cerebrospinal fluid biomarkers are showing promise in aiding early detection.

Treatments for Alzheimer's primarily focus on managing symptoms and may include cholinesterase inhibitors and memantine. However, these treatments are not disease-modifying and offer only modest benefits. Ongoing clinical trials explore potential drug candidates and immunotherapies targeting beta-amyloid and tau to find disease-modifying therapies.

Conclusion

In this review, we have presented some of the causes and possible strategies to treat AD. Several studies have shown that the causal metabolic pathways include extracellular amyloid plaques, intracellular nerve fibers, synaptic degeneration, and neuronal death, ultimately leading to AD as a neurodegenerative disorder. About 70% of AD risk at any age is due to genetics. The most common genetic risk factor for AD is the epsilon 4 allele of the apolipoprotein E gene. Besides the genetic and molecular level, another cause of AD seems to be a dietary lack of vitamin D, whose active form regulates nerve growth factor. Also in AD, a decrease in glucose metabolism in the brain leads to diabetes, the 3 causes of which are still not clear. Finally, we would like to conclude that biomarkers and stem cell therapy can be new technologies for early diagnosis and treatment of AD.

Acknowledgement

None

Conflict of Interest

None

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